How to Do Echo for Noninvasive Hemodynamic Evaluation of the Patient in the Intensive Care Unit: A Consensus Statement of the Italian Society of Echocardiography and Cardiovascular Imaging

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Abstract

Critically ill patients in the intensive care unit (ICU) require continuous hemodynamic monitoring to guide therapeutic decisions and prevent clinical deterioration. Echocardiography has emerged as a cornerstone for noninvasive hemodynamic assessment, offering real-time, bedside evaluation of key parameters such as venous congestion, pulmonary pressures, left atrial pressure (LAP), systemic vascular resistances, cardiac output, and ventricular–arterial coupling. Systemic venous congestion and right atrial pressure (RAP) can be assessed through inferior vena cava diameter measurement and respiratory variation, with additional accuracy provided by the VeXUS score, which incorporates hepatic, portal, and renal vein Doppler profiles. Internal jugular vein assessment and left ventricular (LV) stroke volume variability further refine RAP estimation. Pulmonary hypertension (PH) and right ventricular dysfunction can be evaluated through echocardiographic markers that differentiate precapillary from postcapillary PH, enabling tailored treatment strategies. In addition, echocardiography is fundamental for detecting right ventricular failure, particularly in PH and cardiogenic shock. LAP and systemic hemodynamics are integral to assessing LV diastolic and systolic dysfunction, which are pivotal in heart failure and cardiogenic shock management. Echocardiography also provides insights into vascular system properties and their interaction with cardiac performance, while lung ultrasound aids in detecting interstitial edema of cardiac origin. As a fast, reliable, and reproducible tool, echocardiography is the gold standard for noninvasive hemodynamic assessment in ICU patients, facilitating prompt and precise therapeutic decisions.

Keywords: Echocardiography, hemodynamic monitoring, intensive care unit, left atrial pressure, noninvasive monitoring, pulmonary hypertension, right ventricular function, venous congestion

Introduction

Critically ill patients in the intensive care unit (ICU) require continuous hemodynamic monitoring to promptly guide therapeutic decisions and prevent further clinical deterioration. In this setting, echocardiography emerges as a fast, reliable, and reproducible diagnostic tool,

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making it particularly suitable for bedside application. It provides a comprehensive evaluation of the patient's hemodynamic status including key parameters such as venous congestion, pulmonary pressures and resistances, left atrial pressure (LAP), systemic vascular resistances, cardiac output (CO), and ventricular-arterial coupling (VAC). Given the complexity of hemodynamic assessment, a simplified stepwise approach to hemodynamic assessment is recommended for rapid bedside use, starting with easily obtainable parameters before progressing to more advanced measurements [Central Illustration].

Venous Congestion and Right Atrial Pressure

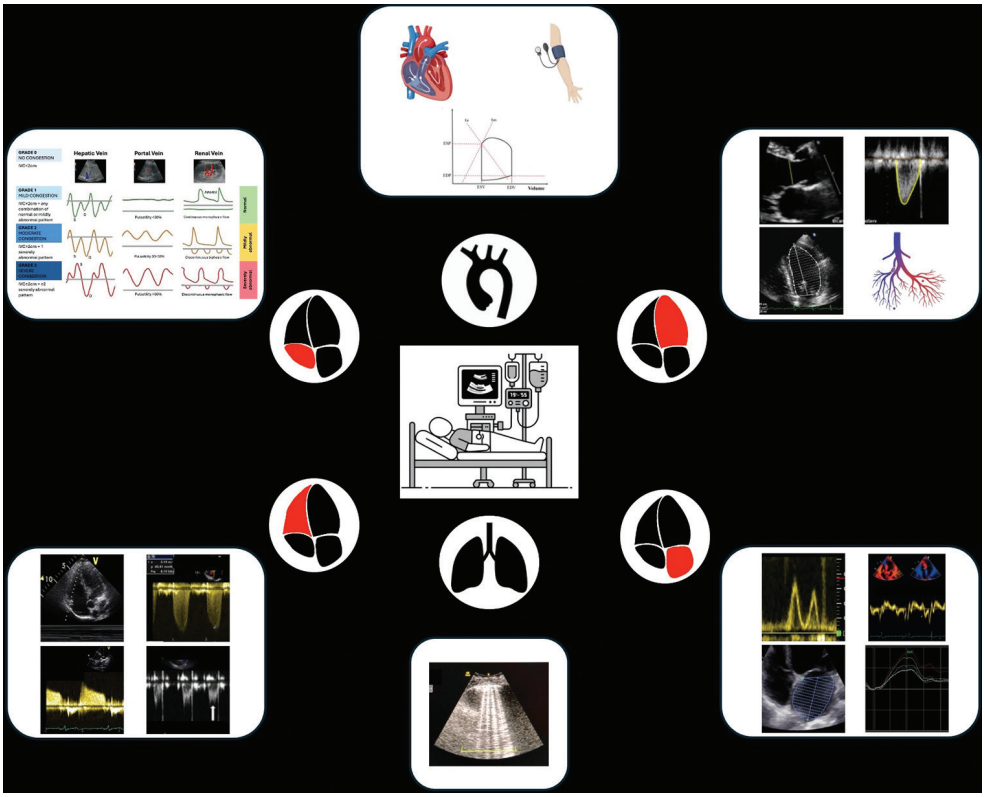
The assessment of systemic venous congestion is essential for the early identification of patients at risk for decompensated heart failure and for evaluating volume status, which informs decisions regarding diuretic or fluid therapy in case of renal failure or hypotension.

Venous congestion and right atrial pressure (RAP) can be evaluated by measuring the inferior vena cava (IVC) diameter and its respiratory changes or, more accurately, by using the VeXUS score, which incorporates IVC diameter and the Doppler profile of hepatic vein (HVen), portal vein (PVen), and renal vein (RVen).[1-3] Additional noninvasive parameters for the RAP estimation include the assessment of the internal jugular vein (IJV) and the respiratory variation of left ventricular (LV) stroke volume (SV).[4,5]

Venous Congestion and Right Atrial Pressure

Inferior vena cava diameter/respiratory collapsibility [Table 1]

- IVC can be visualized using the subxiphoid or lateral transhepatic windows, with measurements taken 0.5–2 cm below its junction with the right atrium
- The caliber variation during an inspiratory sniff is assessed both on B-mode and M-mode, comparing the end-inspiratory and end-expiratory diameters
- RAP can be estimated as follows:
  - ✓ 3 mmHg when IVC ≤2.1 cm and inspiratory collapse is >50%
  - ✓ 15 mmHg when IVC >2.1 cm and inspiratory collapse is <50%
  - ✓ 8 mmHg when IVC >2.1 cm and inspiratory collapse <50% or vice versa
  - ✓ Secondary indices can help refine these estimates, either upgrading to 15 mmHg or downgrading to 3 mmHg:
    - Hepatic vein systolic filling fraction (HVFF) is calculated as the ratio between the velocity-time integral (VTI) of the systolic (S) wave and the sum of the VTIs of the systolic (S) and diastolic (D) waves. An HVFF <55% suggests elevated RAP
    - Tricuspid E/e' ratio: The ratio between peak velocity of the tricuspid E wave (measured on PW-Doppler) to e' (measured on tissue Doppler



Central Illustration: Stepwise echocardiographic assessment of hemodynamics at the bedside. Key parameters include venous congestion, systemic and pulmonary pressures, left atrial pressure, cardiac output, and lung ultrasound findings, following a progression from simple to advanced measurements to guide rapid clinical decision-making

Table 1: Main parameters for noninvasive estimation of right atrial pressure

	Estimated RAP
IVC diameter and respiratory collapsibility	
IVC diameter >2.1 cm + respiratory collapsibility <50%	15 mmHg
IVC diameter <2.1 cm + respiratory collapsibility >50%	3 mmHg
IVC diameter >2.1 cm + respiratory collapsibility >50% or	8 mmHg, or 15 mmHg (when HVFF <55% or tricuspid E/e' >6), or
IVC diameter <2.1 cm + respiratory collapsibility <50%	3 mmHg (when HVFF >55% or tricuspid E/e' <6)
VeXUS score	
No systemic congestion (score=0)	3 mmHg
IVC diameter <2.1 cm	
Mild congestion (score=1)	-
IVC diameter >2.1 cm + one of:	
HVen S/D >1 or >1 (no S reversal)	
PVen continuous flow or PI 30%–50%	
RVen continuous or discontinuous flow (no S reversal)	
Moderate congestion (score=2)	-
IVC diameter >2.1 cm + one of	
HVen S/D <1 or reversed S	
PVen pulsatility >50%	
RVen D only	
Severe congestion (score=3)	>15 mmHg
IVC diameter >2.1 cm + 2 or more of	
HVen S/D <1 or reversed S	
PVen pulsatility >50%	
RVen D only	
Other methods	
RAP=21–24 × HVFF	-
RAP=1.62 × E/e' +2.13	-

E/e'=Ratio between tricuspid E velocity and tricuspid annular e' on tissue Doppler imaging, HVen=Hepatic vein, IVC=Inferior vena cava, HVFF=Hepatic vein systolic filling fraction, PVen=Portal vein, RAP=Right atrial pressure, RVen=Renal vein, PI=Pulsatility index, VeXUS=Venous excess ultrasound score

imaging [TDI]). A tricuspid E/e' >6 indicates elevated RAP.

VeXUS score

- Step 1: Measurement of IVC → when the IVC is <2.1 cm, systemic congestion is very unlikely and can be excluded
- Step 2: Evaluation of HVen, PVen, and RVen (if IVC diameter >2.1 cm)
  - HVen
    - Visualized from the subxiphoid or lateral midaxillary transhepatic windows, enabling the visualization of the right HVen
    - It appears thin-walled and with a blue flow on color-Doppler as it drains into the IVC, moving away from the probe
    - PW-Doppler displays a triphasic or quadriphasic waveform, composed of two anterograde, negative, waves (S, systolic, and D, diastolic) and one or two retrograde, positive, waves (A, atrial contraction; V, between S and D, rebound of the tricuspid annulus to its resting position, after ventricular systole)
    - Patterns
      - Normal → S/D >1
      - Mild congestion → S/D <1
      - Severe congestion → S-wave reversal.
  - PVen
    - Visualized from the subxiphoid or lateral transhepatic windows
    - It has hyperechoic walls, with color Doppler showing blue or red flow depending on the probe's orientation (commonly red flow in the right branch of the PVen when visualized from the midaxillary position)
    - Patterns
      - Normal → continuous flow with no or minimal cyclic variation
      - Mild congestion → pulsatile waveform (which can be quantified by the pulsatility index (PI), calculated as [Vmax-Vmin]/[Vmax] ×100), with a PI = 30%–50%
      - Severe congestion-PI >50%.
  - RVen.
    - The interlobar vein is preferred over the hilar vein due to its alignment with the ultrasound scan plane
    - Best visualized in the lateral midaxillary window at the 10th intercostal space in the patient lying in the left lateral decubitus position. Appears as blue flow on color-Doppler
    - PW displays a monophasic waveform and is visualized together with the arterial vessel which is included in the scan plane above the baseline

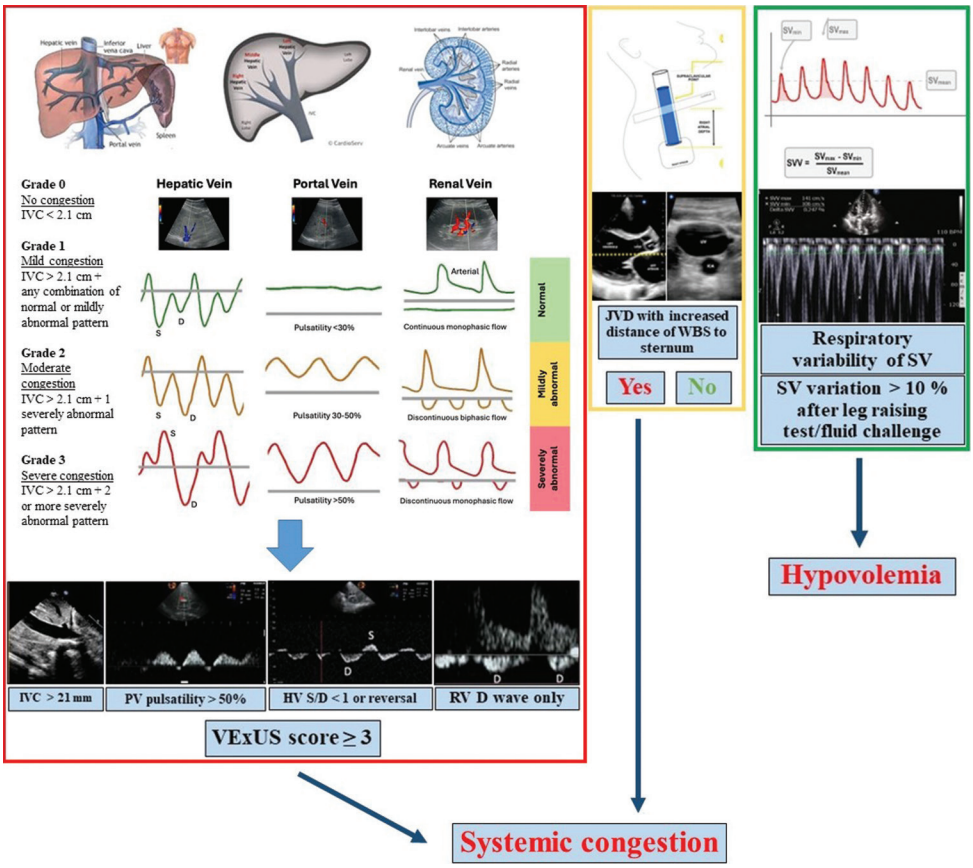


Figure 1: Red box showing, in the top panel, the parameters needed to calculate VeXUS score, and, in the low panel, severely abnormal pattern of each parameter suggestive of severe congestion. Yellow box showing the algorithm for the estimation of central venous pressure by evaluating the internal jugular vein. Green box showing stroke volume variability as a sign of hypovolemia. HVen: Hepatic vein; IVC: Inferior vena cava; JVD: jugular vein distension; PVen: Portal vein; RAP: Right atrial pressure; RVen: Renal vein; SV: Stroke volume

  - Patterns:
    - Normal: Continuous flow
    - Mild congestion: Discontinuous flow with systolic (S) and diastolic (D) waves
    - Severe congestion: D wave only (S waves reversed and hidden in the arterial waveform above the baseline).
- Step 3: Calculation of VeXUS score [Figure 1]
  - Score 0: No systemic congestion (IVC diameter <2.1 cm)
  - Score 1: Mild systemic congestion (IVC diameter >2.1 cm combined with any of normal or mildly abnormal parameters)
  - Score 2: Moderate systemic congestion (IVC diameter >2.1 cm combined with one severely abnormal parameter)
  - Score 3 or more: Severe systemic congestion (IVC diameter >2.1 cm is combined with two or more severely abnormal parameters).

Accessory parameters

Internal jugular vein (IJV)

- Method 1
  - Measure, in patient lying at 45°, the vertical distance from the sternum, at the junction with the clavicle, to the IJV meniscus (the point where the vessel collapses, also called the "wine bottle sign," WBS)
  - Measure the right atrial depth (RAD) from the parasternal long-axis view by taking the depth of the posterior wall of LV tract outflow (LVOT), near the attachment of noncoronary aortic cusp
  - RAP is calculated, in cm H_2O (or converted to mmHg by multiplying by 0.74), as the sum of the vertical distance to WBS and RAD.
- Method 2
  - Measure the IJV caliber variation during the Valsalva maneuver
  - A Valsalva/rest diameter ratio <1.6 predicts RAP >7 mmHg.

Left ventricular stroke volume variation (SVV)

- Evaluation of variation of LVOT-VTI, on PW-Doppler, during
  - Respiratory efforts
  - Fluid challenge
  - Passive leg raising.
- An increase in LVOT VTI >10% suggests hypovolemia
- Limitations in ICU patients
  - Arrhythmias: The presence of atrial fibrillation or frequent ectopic beats can significantly alter

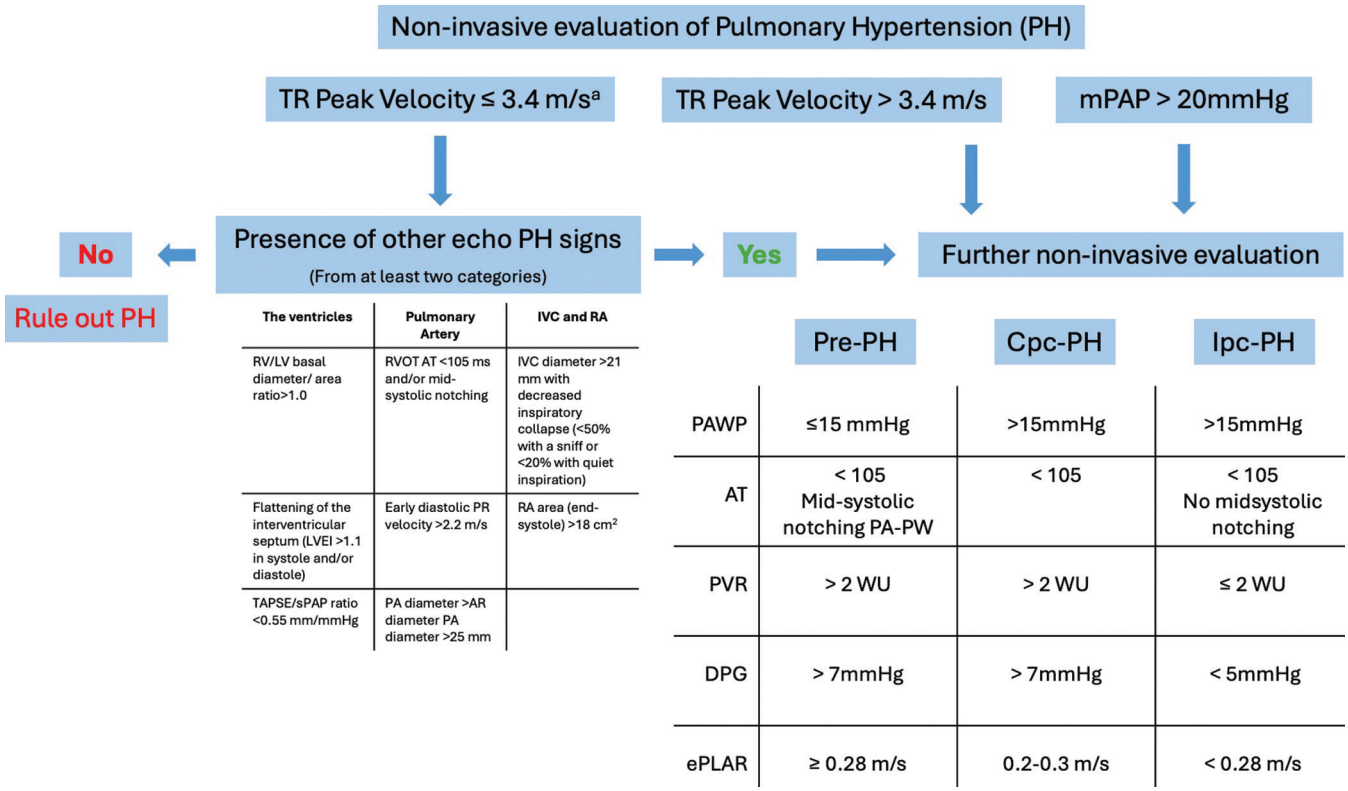


Figure 2: Noninvasive evaluation of pulmonary hypertension. Starting from the evaluation of tricuspid regurgitation peak velocity or mean pulmonary artery pressure, this approach allows for a noninvasive assessment based on echocardiographic parameters to distinguish between precapillary, postcapillary, or combined pulmonary hypertension. AR: Aortic root; Cpc-PH: Combined post- and precapillary pulmonary hypertension; DPG: Diastolic pressure gradient; ePLAR: Echocardiographic pulmonary-to-left atrial ratio; Ipc-PH: Isolated postcapillary pulmonary hypertension; IVC: Inferior vena cava; LVEI: Left ventricle eccentricity index; LV: Left ventricle; mPAP: Mean pulmonary artery pressure; PA: Pulmonary artery; PA-PW: Pulmonary artery pulsed-Doppler wave; PAWP: Pulmonary arterial wedge pressure; PR: Pulmonary regurgitation; Pre-PH: Precapillary pulmonary hypertension; PVR: Pulmonary vascular resistance; RA: Right atrium; RV: Right ventricle; RVOT AT: Right ventricle outflow tract acceleration time; sPAP: Systolic pulmonary arterial pressure; TAPSE: Tricuspid annular plane systolic excursion; TR: Tricuspid regurgitation; WU: Wood units. [a]Or unmeasurable

beat-to-beat SV, making SVV measurements unreliable. In these cases, alternative hemodynamic assessments may be more appropriate

- Low-tidal volume ventilation: SVV is most accurate in patients receiving controlled mechanical ventilation with tidal volumes ≥8 mL/kg. In patients on lung-protective ventilation strategies (e.g., acute respiratory distress syndrome [ARDS] with tidal volumes <6 mL/kg), SVV may underestimate fluid responsiveness, necessitating the use of dynamic tests like passive leg raising or end-expiratory occlusion maneuvers.

Pulmonary and Right Ventricular Hemodynamics

Echocardiography is the first-line diagnostic method for identifying pulmonary hypertension (PH), although definitive confirmation requires invasive right heart catheterization. In this clinical context, several noninvasive parameters can help differentiate between precapillary and postcapillary forms of PH, leading to different therapeutic strategies.[1,6,7] Moreover, echocardiography plays a critical role in detecting the onset of right ventricular failure, across a range of clinical scenarios, including PH and cardiogenic shock.[8,9]

The evaluation of pulmonary and right ventricular hemodynamics relies on several key parameters, detailed as follows.

Pulmonary and right ventricular hemodynamics [Figure 2]

- Systolic pulmonary artery pressure (sPAP)
  - sPAP = 4 × (peak velocity of tricuspid regurgitation [TR]^2) + RAP.
- Mean pulmonary artery pressure (mPAP)
  - Method 1 → mPAP = 4 × (peak velocity of pulmonary regurgitation [PR]^2) + RAP
  - Method 2 → mPAP = 1/3× (sPAP) +2/3 × (diastolic pulmonary artery pressure [dPAP])
  - Method 3 → Derived from VTI of TR
  - Method 4 → Using pulmonary velocity acceleration time (AT) with the formula 79 × (0.45 × AT) or when AT is <120 ms: 90 × (0.62 × AT), based on the inverse relation between pulmonary pressures and the AT.
- dPAP
  - dPAP = 4 × (end-diastolic velocity of pulmonary regurgitation [PR]^2) + RAP
  - In the absence of precapillary PH, dPAP approximates LAP or pulmonary artery wedge pressure (PAWP)

- When the difference between dPAP and noninvasively estimated PAWP (diastolic pressure gradient or DPG) exceeds 7 mmHg, a combination of pre- and post-capillary PH can be suspected.

- AT (pulmonary artery AT):
  - Measured from the onset of the QRS to the onset of pulmonary peak flow
  - Inverse relation with pulmonary pressures
  - AT <105 ms suggests PH
  - Midsystolic notching of pulmonary artery flow indicates PH and increased pulmonary vascular resistances.
- Tricuspid regurgitation peak velocity (TrV):
  - *High probability* of PH: TrV >3.4 m/s, or 2.8–3.4 m/s with at least two echocardiographic signs from different categories
    - Ventricles category
      - RV/LV basal ratio >1
      - Septal flattening with an LV eccentricity index >1.1
      - Tricuspid annular plane systolic excursion (TAPSE)/sPAP <0.55.
    - Pulmonary artery category
      - AT <105 ms
      - Early PR velocity >2.2 m/s
      - Pulmonary artery diameter >25 mm.
    - IVC and RA category.
      - Right atrial area >18 cm^2 at end-systole.
      - IVC diameter >21 mm with inspiratory collapsibility <50%.
  - *Intermediate probability* of PH: TrV ≤2.8 m/s with at least two echocardiographic signs from different categories, or TrV 2.8–3.4 m/s without echo signs
  - *Low probability* of PH when ≤2.8 m/s without echo signs.
- Pulmonary vascular resistances
  - PVR = (TRV/TVI of right ventricular outflow tract [RVOT]) × 10 + 0.16, by Abbas *et al*.,[10] based on the assumption that resistance = pressure gradient/flow and measuring the peak velocity of TR in m/s and the RVOT VTI in cm
  - Helps to differentiate PH caused by high flow versus increased vascular resistances, as seen in idiopathic pulmonary arterial hypertension, lung diseases, or hypoxic vasoconstriction.
- Pulmonary arterial compliance (PAC)
  - PAC = RV SV/pulmonary pulse pressure
  - PAC decreases earlier than PVR in PH, with a hyperbolic relation between PAC and PVR
  - PAC is a better predictor of mortality following acute pulmonary embolism than PVR.
- Echocardiographic pulmonary-to-left atrial ratio (ePLAR)
  - ePLAR = TR peak velocity/(mitral E velocity/septal e')
  - Differentiates between precapillary and postcapillary PH

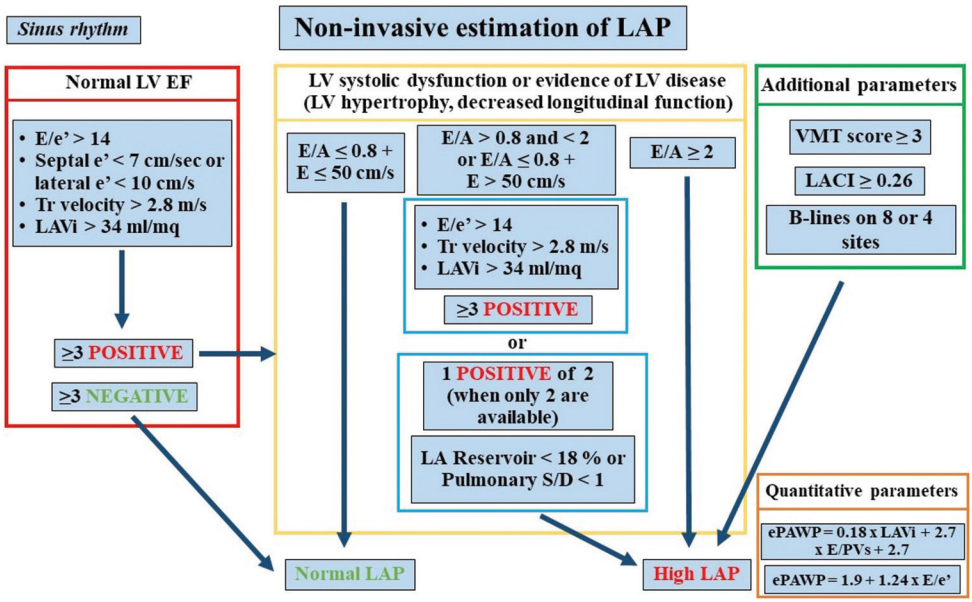

Sinus rhythm
Non-invasive estimation of LAP
Normal LV EF
• E/e' > 14
• Septal e' < 7 cm/sec or lateral e' < 10 cm/s
• Tr velocity > 2.8 m/s
• LAVi > 34 ml/mq
≥3 POSITIVE
≥3 NEGATIVE
LV systolic dysfunction or evidence of LV disease (LV hypertrophy, decreased longitudinal function)
E/A ≤ 0.8 + E ≤ 50 cm/s
E/A > 0.8 and < 2 or E/A ≤ 0.8 + E > 50 cm/s
E/A ≥ 2
• E/e' > 14
• Tr velocity > 2.8 m/s
• LAVi > 34 ml/mq
≥3 POSITIVE
or
1 POSITIVE of 2 (when only 2 are available)
LA Reservoir < 18 % or Pulmonary S/D < 1
Additional parameters
VMT score ≥ 3
LACI ≥ 0.26
B-lines on 8 or 4 sites
Quantitative parameters
ePAWP = 0.18 x LAVi + 2.7 x E/PVs + 2.7
ePAWP = 1.9 + 1.24 x E/e'
Normal LAP
High LAP

Figure 3: Red box showing the algorithm, in sinus rhythm, for the evaluation of left ventricle (LV) diastolic function in patients with normal LV ejection fraction and without additional signs of LV disease. Yellow box showing the algorithm for the evaluation of LV diastolic function in patients with LV systolic dysfunction and/or pathologic remodeling. Green box showing additional parameters for the estimation of left atrial pressure (LAP). Orange box showing parameters to, numerically, quantify LAP and pulmonary artery wedge pressure. E/A: ratio between peak velocities of mitral E and A; E/e' ratio between mitral E velocity and mean e' on tissue Doppler imaging; ePAWP: Estimated pulmonary artery wedge pressure; LACI: Left atrial coupling index; LAP: Left atrial pressure; LA Reservoir, two-dimensional strain of atrial walls during reservoir phase; LAVi: Left atrial volume index; Tr: Tricuspid regurgitation; VMT: Visually assessed time difference between mitral valve and tricuspid valve opening

- An ePLAR value ≥0.3 m/s suggests precapillary PH, while a value <0.2 m/s is indicative of postcapillary PH.

- Pulmonary artery pulsatility index (PAPi)
  - PAPi = (sPAP-dPAP)/RAP
  - Reflects RV systolic performance, preload, and afterload, since pulse pressure depends on both SV and arterial compliance, while RAP measures preload.
- Right ventricular stroke work index (RVSWI)
  - Method 1 → RVSWI = (mPAP − RAP) × RV SV index (SVi) × 0.0136 (derived invasively from RV pressure-volume loop)
  - Method 2 → RVSWI = TR peak velocity (or VTI)/RV SVi, being Tr peak velocity and VTI, respectively, an indirect measure of RV systolic and mean intraventricular pressure
  - Reflects overall RV systolic performance.
- TAPSE/sPAP
  - Represents RV-to-PA coupling and normalize RV systolic function with afterload
  - Correlates well with invasively measured PVR and is a prognostic marker, particularly in pulmonary arterial hypertension.

Left Atrial Pressure and Systemic Hemodynamics

Echocardiography allows the identification of LV diastolic dysfunction that can be associated with increased LAP, as well as systolic dysfunction, commonly observed in patients with heart failure and cardiogenic shock. These are essential data to guide therapeutic decision-making.[11-15] Furthermore, echocardiography facilitates the assessment of arterial system properties and their interaction with LV performance, offering insights into the dynamic interplay between the heart and vascular system.[16-21] In addition, lung ultrasound is a valuable tool for the detection of interstitial edema of cardiac origin.[22]

Several parameters for assessing LAP and systemic hemodynamics are outlined as follows.

Left atrial pressure estimation

- Method 1 [sinus rhythm, Figure 3]
  - Step 1 → evaluation of LV ejection fraction (LVEF) and other parameters, such as LV geometry, myocardial mass, and systolic longitudinal function, that may suggest cardiac disease
  - Step 2 → when LV EF is normal and there are no signs of cardiac disease, the presence of three or more positive criteria from the following indicates LV diastolic dysfunction
    - TDI septal <7 cm/s and lateral e' <10 cm/s
    - Left atrial volume index (LAVi) >34 mL/m^2
    - TR peak velocity >2.8 m/s
    - Mean E/e' >14.
  - Step 3 → when 3 or more positive criteria of the above are present, or, when LV EF is lower than normal or other signs of cardiac disease are found (LV hypertrophy, impaired global longitudinal strain, etc.), E/A ratio is, firstly, measured:
    - E/A <0.8 + E-wave <50 cm/s → *Grade I diastolic dysfunction*
    - E/A between 0.8 and 2 or E/A <0.8 + E-wave >50 cm/s → Tr peak velocity, LAVi and E/e' must be evaluated:

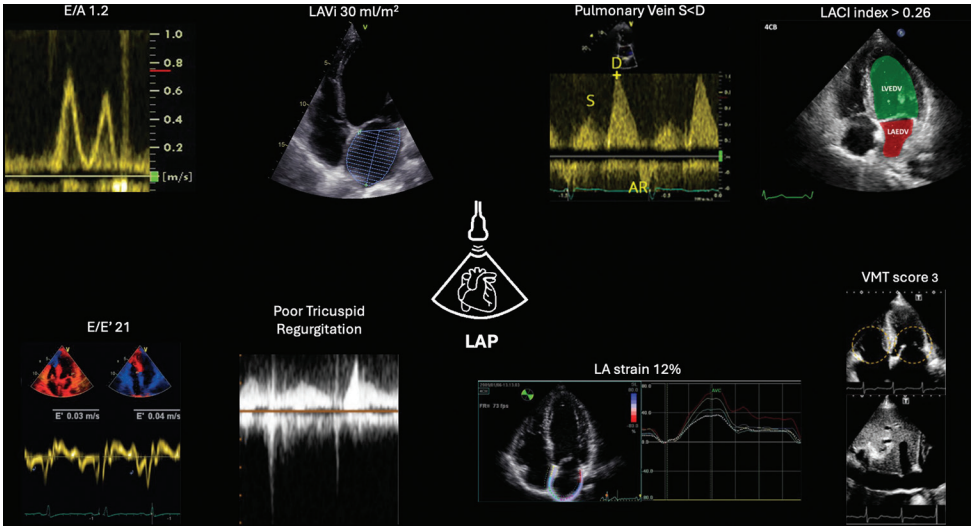


Figure 4: Example of left atrial pressure estimation in patients with depressed ejection fraction and left ventricle disease, when E/A is between 0.8 and 2 and only two criteria are available (average E/e', tricuspid regurgitation velocity, and left atrial volume index). In cases where one criterion is positive and another is negative, left atrial reservoir strain can help distinguish between Grade 1 and Grade 2 diastolic dysfunction. In addition, two alternative methods for estimating filling pressures are illustrated: the left atrioventricular coupling index and the visually assessed time difference between mitral valve and tricuspid valve opening (VMT), combined with inferior vena cava size. IVC: Inferior vena cava; LA: Left atria; LACI: Left atrioventricular coupling index; LAP: Left atrial pressure; LAVi: Left atrial volume index; VMT: Visually assessed time difference between mitral valve and tricuspid valve opening

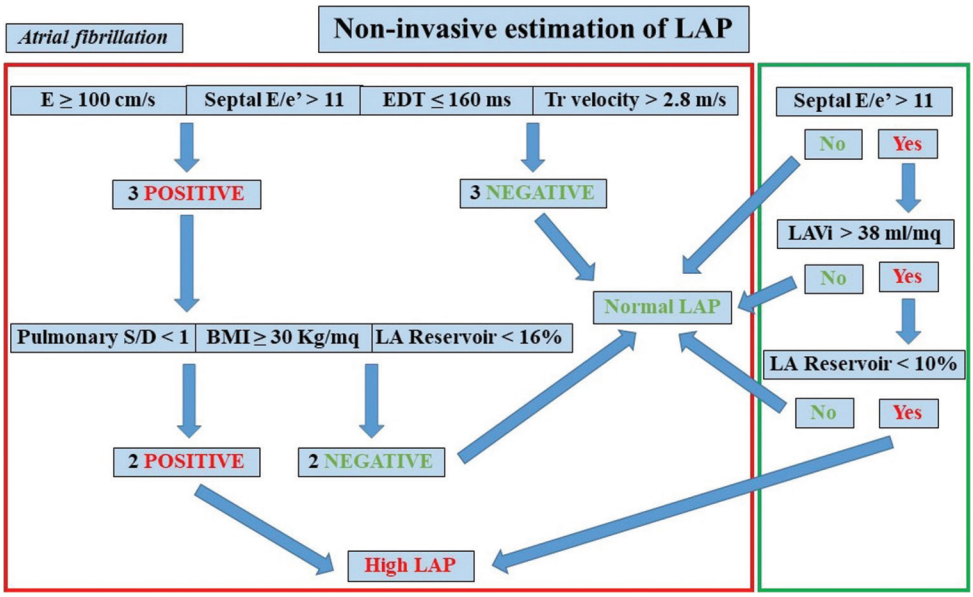


Figure 5: Red box showing the algorithm, in atrial fibrillation, for the evaluation of left atrial pressure (LAP), by using step 1 composed of four parameters, such as mitral E peak velocity, septal E/e', mitral E deceleration time, Tr velocity, followed, when needed, by a step 2, composed of three parameters, such as pulmonary S/D, body mass index and left atria (LA) Reservoir strain. Green box showing a stepwise algorithm, in atrial fibrillation, for the estimation of LAP, composed of septal E/e', left atrial volume index, and LA Reservoir strain. BMI: Body mass index; E/e' ratio between mitral E velocity and septal e' on tissue Doppler imaging; LAP: Left atrial pressure; LA Reservoir, two-dimensional strain of atrial walls during reservoir phase; LAVi: Left atrial volume index; Pulmonary S/D, ratio between peak velocities Tr, tricuspid regurgitation

- If 2 or more of the criteria are positive (with reference to the mentioned cutoff values), *Grade II diastolic dysfunction* can be postulated, otherwise Grade I.

When only 2 of the abovementioned criteria are available and only 1 of 2 criteria is positive, the degree of diastolic dysfunction cannot be determined. In such cases, LA reservoir strain provides additional diagnostic value [Figure 4]. LA reservoir strain is evaluated using two-dimensional speckle-tracking echocardiography, which tracks the deformation of the left atrial wall throughout the cardiac cycle. Acquisition is performed in the apical four-chamber and two-chamber views, focusing on the atrial walls during the reservoir phase (from the onset of ventricular systole to the peak of the P-wave). The strain curve is derived by analyzing the displacement of speckles on the left atrial myocardium. Peak reservoir strain (the maximal deformation during systole) is measured, with values <18% suggesting impaired diastolic function. For higher sensitivity, values <10% are strongly associated with elevated LAP. This technique provides a load-independent assessment of LA function, aiding in the differentiation between Grade I and Grade II diastolic dysfunction.

- E/A >2 → *Grade III diastolic dysfunction.*

- Method 2 [Atrial fibrillation, Figure 5]
  - *Algorithm 1*
    - Step 1 → measurement of four parameters, which suggest increased LAP when 3 or more are positive (3 or more negative exclude increased LAP):
      - mitral E velocity, positive when ≥100 cm/s
      - septal E/e', positive when >11
      - E deceleration time, positive when equal or <160 ms
      - Tr peak velocity, positive when >2.8 m/s and
    - Step 2 → (*when step 1 nondiagnostic*) measurement of three parameters, which suggest increased LAP when 2 criteria of 3 are positive (or exclude increased LAP when 2 of 3 are negative):
      - LA reservoir strain, positive when <16%
      - pulmonary venous S/D ratio, positive when <1
      - BMI positive when ≥30 kg/mq
  - *Algorithm 2*
    - Step 1 → septal E/e' ≥11 (if negative stop, if positive go further)
    - Step 2 → LAVi ≥38 mL/m^2 (if negative stop, if positive go further)
    - Step 3 → LA reservoir strain <10% (if positive, suggestive of increased LAP).
- Method 3
  - Left atrioventricular coupling index is calculated as the ratio between LA minimum volume and LV end-diastolic volume, with a cutoff value of ≥0.26.
- Method 4
  - VMT score is based on the time difference in opening of tricuspid (TV) and mitral (MV) valves
    - 0 → when TV opens first
    - 1 → when TV and MV open simultaneously
    - 2 → when MV opens first
    - 3 → when MV opens first + IVC diameter >21 mm

with inspiratory collapsibility <20%.
    - A VMT score ≥2 has been shown to be associated with increased LAP.
- Other methods
    - LAP can be also quantitatively assessed:
        - 1.9 + 1.24 × E/e'
        - 0.18 × LAVi + 2.7 × E/PVs (systolic peak velocity of pulmonary vein flow) + 2.7.

LAP is increased when is >15 mmHg.

B-lines

- B-lines are vertical, laser-like, respiratory-synchronous reverberation artifacts that originate from the pleural line, which appears hyperechoic and exhibits phasic movement with respiratory efforts (commonly referred to as "lung sliding"). These artifacts extend to the bottom of the

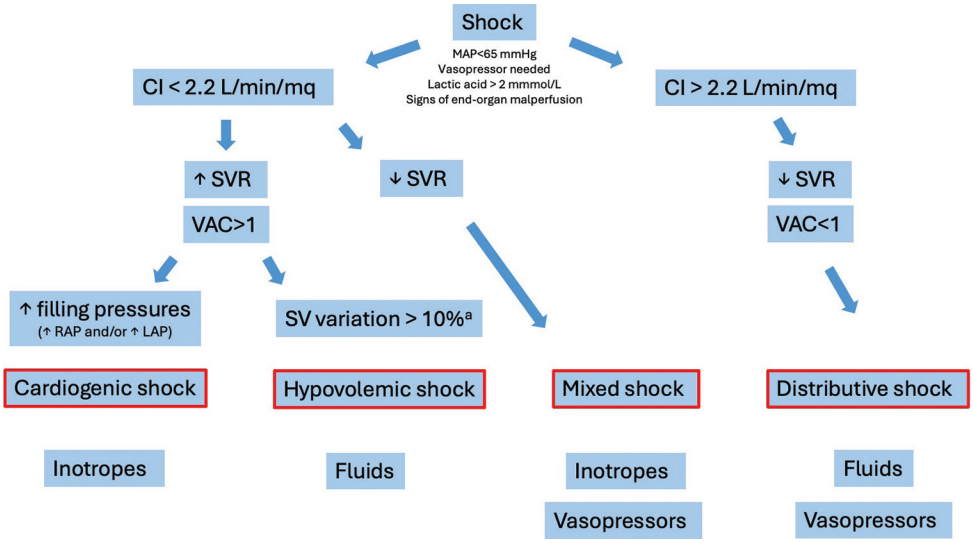


Figure 6: Critical care ultrasound in shock. The proposed algorithm allows the classification of different types of shock based on echocardiographic parameters. CI: Cardiac index; LAP: Left atrial pressure; MAP: Mean arterial pressure; RAP: Right atrial pressure; SV: Stroke volume; SVR: Systemic vascular resistances; VAC: Ventricular–arterial coupling; [a]after leg raising test/fluid challenge

Table 2: Main parameters for the noninvasive hemodynamic study

	How to calculate	Normal/(reference) values
sPAP	4 × TrV^2 + RAP	15–30 mmHg
dPAP	4 × proto-diastolic PrV^2 + RAP	8–20 mmHg
mPAP	4 × end-diastolic PRV^2 + RAP	4–12 mmHg
PVR	TRV/TVI RVOT × 10 + 0.16	<2 WU (<0.2)
PAC	RV SV/PP	<3 mL/mmHg
ePLAR	TrV/(mitral E wave/septal e')	(<0.28 m/s: Postcapillary PH)
PAPi	(sPAP-dPAP)/RAP	(sPAP- Severe RV failure)
RVSWI	4 × TrV^2/RV SVi or Tr VTI/RV SVi	>8–12 mmHg*mL/m^2
TAPSE/sPAP	TAPSE/(4 × TrV^2 + RAP)	(<0.55 suggestive of PH; <0.35 mm/mmHg: RV to PA uncoupling and RV failure)
CO	CO=(π/4 × LVOT D^2) × VTI × heart rate	CO=4–8 L/min
CI	CI=CO/BSA	CI=2.5–4 L/min/m^2
CPO	([MAP-RAP] × CO)/451	<0.6 W/min is associated with worse prognosis in cardiogenic shock
SVR	(MAP-RAP)/CO × 80	900–1200 dynes/s/cm^{-5} (11–15 WU, when applying the formula without multiplying for 80)
	MRV/LVOT VTI	(MRV/VTI LVOT >0.27 suggests SVR >14 WU)
Ea	Ea=ESP*/SV	Ea=2.2±0.8 mmHg/mL
Ev	Ev=ESP*/ESV	Ev=2.3±1 mmHg/mL
VAC	VAC=Ea/Ev	VAC=1±0.36 mL

*Calculated from brachial systolic pressure ×0.9. CO=Cardiac output, CI=Cardiac index, CPO=Cardiac power output, dPAP=Diastolic pulmonary artery pressure, Ea=Arterial elastance, Ev=Ventricular elastance, ePLAR=Pulmonary left-to-atrial ratio, mPAP=Mean pulmonary artery pressure, PAC=Pulmonary artery compliance, PAPi=Pulmonary artery pulsatility index, PVR=Pulmonary vascular resistances, sPAP=Systolic pulmonary artery pressure, RVSWI=Right ventricular stroke work index, SVR=Systemic vascular resistances, TAPSE/sPAP=Ratio between tricuspid annular plane systolic excursion and systolic pulmonary artery pressure, VAC=Ventricular-arterial coupling, MRV=Mitral regurgitation peak velocity, VTI=Velocity-time integral, LVOT=Left ventricular tract outflow, ESP=End-systolic pressure, ESV=End-systolic volume, SV=Stroke volume, MAP=Mean arterial pressure, RAP=Right atrial pressure, BSA=Body surface area, RV=Right ventricle, PA=Pulmonary artery, TRV=Tricuspid regurgitation peak velocity, TVI=Time-velocity integral, RVOT=Right ventricle outflow tract, WU=Wood units, SVi=Stroke volume index, PP=Pulse pressure, PRV=Pulmonary regurgitation velocity

ultrasound scansion sector and are caused by decreased air content due to conditions such as edema or fibrosis
- Scanning protocols (using phase-array or convex transducers)
  - *8-zone* protocol: Each hemithorax is divided into four areas from the parasternal to posterior axillary lines: upper and lower anterior chest areas and upper and basal lateral chest areas
  - *4-zone* protocol: Only four chest areas are assessed, corresponding to the third intercostal space along the anterior and midaxillary lines.
- Assessment technique: Once the appropriate transducer is selected, the clinician performs the ultrasound in a linear, stepwise fashion across the designated zones. The pleural line is closely observed during both inspiration and expiration. B-lines are identified when there is a hyperechoic vertical artifact extending from the pleural line, consistent with lung sliding. The presence of more than three B-lines in a single zone is considered positive for potential pulmonary congestion or interstitial disease
- While B-lines are a hallmark of cardiogenic edema, they are not highly specific and can be observed in conditions such as ARDS, interstitial pneumonia, and pulmonary fibrosis. Distinguishing between cardiogenic and noncardiogenic causes is critical, as B-lines in ARDS or fibrosis may present with a more diffuse, heterogeneous distribution and be accompanied by an irregular, thickened pleural line, in contrast to the more homogeneous, gravity-dependent distribution seen in cardiogenic edema
- Limitations in ICU patients: The evaluation of pleural and lung artifacts can be challenging in critically ill patients due to the factors such as mechanical ventilation, subcutaneous emphysema, and pleural effusions, which may obscure typical findings. In addition, lung ultrasound findings should always be interpreted in the clinical context to avoid misclassification of noncardiogenic pulmonary pathology as cardiogenic edema
- Cardiac B-lines are typically homogeneous, gravity related in distribution, regular in appearance, and associated with normal sliding pleural movement. Noncardiogenic B-lines (e.g., in ARDS or fibrosis) tend to have an irregular appearance, a nongravity-related distribution and are associated with thickened, irregular pleural line
- Quantification methods:
  - Score-based method: A minimum of three B-lines per chest area is required to classify it as a positive zone. The total number of positive zones is then summed
  - Count-based method: Each B-line is counted and summed individually. When B-lines are confluent, their number is estimated by dividing the percentage of occupied space by 10.

Systemic and Left Ventricular Hemodynamic Parameters [Figure 6 and Table 2]

- CO and cardiac index (CI)
  - CO: calculated as LVOT cross-sectional area ($\pi/4 \times D^2$) × LVOT VTI × heart rate, based on circular geometric assumption and applying the continuity equation. LVOT diameter is measured in the parasternal long axis at mid-systole from the inner edge to inner edge at the junction of the aortic leaflets to the interventricular septum and mitral-aortic intervalvular fibrosa. LVOT VTI is assessed using PW-Doppler in the apical 5-chamber view, at the same level
  - CI: derived by dividing CO by the body surface area.
- Cardiac power output (CPO)
  - CPO = [(MAP-RAP) × CO]/451: the constant (451) is derived from assumed normal values: RAP = 3 mmHg, MAP = 93.3 mmHg (based on the values of systolic and diastolic blood pressure, respectively, of 120 mmHg and 80 mmHg) and of CO = 5.0 l/min. This results in a cardiac work/minute of 1.0 W/min, considered the normal value
  - CPO reflects the cardiac work performed per unit time and is one of the strongest prognostic indictors in patients with cardiogenic shock.
- SVR (systemic vascular resistances):
  - Method 1 → SVR = (MAP-RAP)/CO × 80
  - Method 2 → SVR = mitral regurgitation peak velocity (MRV)/LVOT-VTI
  - SVR reflects the interaction between flow (i.e., CO) and the arterial system properties, such as vascular tone, which can be increased in hypovolemic, obstructive, and cardiogenic shock or decreased in distributive (i.e., septic) shock.
- Arterial elastance (Ea), ventricular elastance (Ev), and VAC (arterial and ventricular elastance, VAC):
  - Ea: Reflects arterial system properties, including vascular tone and stiffness; calculated as end-systolic pressure divided by SV (ESP/SV). ESP is calculated from brachial systolic pressure × 0.9
  - Ev: Reflects load-independent LV contractility; it is calculated from
    - Method 1 → Ev = ESP divided by end-systolic volume (ESP/ESV)
    - Method 2 (by Chen *et al*.[23]) → Ev = [Diastolic blood pressure (DBP) − (End (est) × Systolic blood pressure (SBP) × 0.9)]/[End (est) × SV], where End (est) is a constant representing normalized ventricular elastance at the onset of ejection. This value can be derived using automated tools, requiring the measurement of LVEF and the preejection time on LVOT PW-Doppler.
  - VAC: calculated as Ea/Ev or, more simply, ESV/SV (the simplified approach is, however, limited by the fact that ESV and SV are both related to LVEF

and, thus, the so-derived VAC does not provide additional information with respect to LV EF)
  - VAC reflects the interaction between LV ejection and the subsequent changes in arterial pressure, reflecting global cardiovascular performance and efficiencyo Ventricular–arterial uncoupling occurs when Ea and Ev do not show a linear relation, with Ea higher or lower than Ev, such occurring, respectively, in hypertensive emergency (VAC >1) and septic shock (VAC <1) or when Ev is more impaired than Ea as can be observed in cardiogenic shock (VAC >1)
  - In these clinical settings, different therapeutic implications emerge, such as vasodilators, when Ea is disproportionately high relative to Ev, vasopressors when Ea is decreased, inotropes when Ev is impaired, and inodilators when decreased Ev is combined with increased Ea.

Troubleshooting

Obtaining high-quality ultrasound images in critically ill patients can be challenging due to patient instability, mechanical ventilation, suboptimal acoustic windows, or ICU-specific factors such as edema and surgical dressings. The following strategies can help optimize imaging:[24]

- Choose the Right Transducer and Preset: Use a phased-array probe for cardiac and lung imaging, a curvilinear probe for abdominal and IVC assessments, and adjust presets accordingly (e.g., cardiac, FAST, or abdominal)
- Optimize Depth and Gain: Adjust depth to focus on the region of interest, increase gain for better visualization in hypoechoic areas, and fine-tune time gain compensation sliders to enhance contrast
- Utilize Image Enhancement Features: Apply auto-optimization to enhance contrast, and use Zoom for better resolution when magnifying structures
- Improve Patient Positioning: If possible, adjust patient positioning – left lateral tilt for better cardiac windows, head elevation for IVC imaging, and lung ultrasound adjustments based on air and fluid distribution (e.g., pneumothorax in nondependent areas, effusions in dependent areas)
- Adapt to Mechanical Ventilation and Artifacts: Be mindful of respiratory-induced motion, use M-mode to confirm findings (e.g., seashore sign for pneumothorax), and differentiate between true pathology and artifacts such as acoustic shadowing and enhancement
- Consider Alternative Windows: If transthoracic echocardiography (TTE) is inadequate, use the subcostal window or switch to transesophageal echocardiography (TEE) for superior cardiac imaging in ventilated patients
- Minimize External Interference: Reduce ambient lighting, ensure adequate gel application, and adjust line density settings to balance resolution and frame rate for dynamic cardiac assessments.

By implementing these techniques, clinicians can optimize ultrasound imaging in the ICU, ensuring accurate hemodynamic assessments despite challenging conditions.

Conclusions

Echocardiography is the method of choice for the noninvasive, comprehensive hemodynamic evaluation of critically ill patients, particularly in the cardiac ICU. It is the only technique that allows for a complete assessment of cardiovascular status at the bedside, providing real-time information on cardiac function, volume status, and pulmonary circulation. While echocardiography offers invaluable insights, it should not be used in isolation. Instead, it should be integrated into a multimodal monitoring strategy that includes clinical assessment, invasive measurements when necessary, and additional imaging and functional tests. Moreover, for a comprehensive hemodynamic evaluation, an integration with other noninvasive tools (e.g., passive leg raising and bioimpedance) could be necessary, in some cases. By combining echocardiographic data with systemic hemodynamic indices, lung ultrasound, and dynamic assessments like fluid responsiveness tests, clinicians can obtain a holistic view of a patient's cardiovascular performance. This integrated approach is especially critical in complex shock states, where accurate differentiation of causes and precise, individualized interventions can dramatically influence patient outcomes.

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Conflicts of interest

There are no conflicts of interest.

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